Section of Neurology.

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Spontaneous Sub-arachnoid Hæmorrhage.

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THE title of my paper calls in the first instance for a word of explanation. From a study of the literature it seems that the term "spontaneous" in this connexion has acquired for some persons a mystical significance, and is conferred by them upon those cases of sub-arachnoid hæmorrhage for which no apparent cause is to be found either at the bedside or after death. *Omne ignotum pro magnifico!* Let us admit the existence of many cases, the origin of which remains obscure, and suffer the impatience of our present ignorance without seeking refuge behind a formula.

The term "spontaneous," as I interpret it here, covers all cases of sub-arachnoid hæmorrhage of origin other than traumatic. The word is used improperly, but I must refer you as precedent for the distinction to one of the earliest papers upon the subject published by Sir Samuel Wilks in 1859 and entitled "Sanguineous Meningeal Effusion (Apoplexy): Spontaneous and from Injury."[1]

Blood appearing in the sub-arachnoid space may obtain entrance to it by various channels. It may be derived from the rupture of vessels lying within the sub-arachnoid space itself. It may find its way into the sub-arachnoid space from hæmorrhage into the subdural cavity (as commonly occurs in traumatic lesions of the veins passing from the cerebral cortex into the great sinuses).

Or again, blood derived from the rupture of a vessel within the nervous substance is not infrequently discharged into the sub-arachnoid space either by breaking through the pia mater if the hæmorrhage be superficial, or, more usually, since cerebral hæmorrhage is, as a rule, deeply situated, the effusion may burst into one of the ventricles and make its way into the general sub-arachnoid cavity by the channels which connect this with the ventricular system.

Of these three sources of sub-arachnoid hæmorrhage I propose only to discuss the first and third, since, so far as I am aware, subdural hæmorrhage does not occur except as the result of injury, and is therefore excluded from present consideration.

The clinical evidence of sub-arachnoid hæmorrhage may be considered under two heads, first that derived from the examination of the cerebro-spinal fluid, second that which may be obtained by observation of the history, symptoms and course of the illness.

The changes which occur in the cerebro-spinal fluid in this condition were fully described by Froin in his thesis of 1904, and there is little to add to his original observations.

Shortly after the onset of the hæmorrhage the fluid obtained by lumbar puncture appears on withdrawal to be mixed with blood, and is usually under increased pressure.

[1] *Guy's Hosp. Rep.*, 1859, 3rd ser., v, p. 119.

If this fluid be collected in a series of three test tubes, the same degree of admixture is to be observed in each. The proportion of blood to cerebro-spinal fluid will vary on the one hand according to the severity of the leakage, and on the other hand according to the situation of the ruptured vessel. Blood extravasated into one of the basal cisterns will find its way more readily to the site of lumbar puncture than an effusion originating from the surface of one of the cerebral hemispheres, or from the wall of one of the lateral ventricles. Thus the colour of the fluid when first withdrawn may vary from an opalescent pink to that of pure blood—the highest count of red cells per c.mm. which I have found recorded being 3,380,000.

On being allowed to stand in the test-tube this sanguineous fluid does not form any coagulum. The red cells sink to the bottom of the tube, and the clear supernatant fluid appears coloured by the presence of altered blood pigments. This colour varies from rose-pink or brown to golden yellow. Subsequently it undergoes changes together with the other elements in the fluid, which I shall refer to later.

Thus the distinctive macroscopic features of the cerebro-spinal fluid in sub-arachnoid hæmorrhage are three:—

(1) An even admixture of blood which is the same in a series of specimens collected at the same puncture.

(2) Absence of coagulum.

(3) Pink, brown or yellow coloration of the clear supernatant fluid, which is apparent when the red cells have been allowed to sink to the bottom of the tube.

None of these three features is to be observed in cerebro-spinal fluid contaminated by the accidental injury of a vein during lumbar puncture. Here, on the other hand, when the fluid is obtained in a series of tubes, the first is seen to be more deeply stained than the last. On standing in the test-tube the fluid forms a coagulum, and the clear fluid in the upper portion of the tube remains colourless.

The explanation offered by Froin for these differences appears to be satisfactory. In sub-arachnoid hæmorrhage the effusion of blood forms a coagulum at and around the site of the leakage. The proportion of red cells entangled in this coagulum varies somewhat according to the severity of the leakage and its situation. If the latter be in one of the basal cisterns where the circulation of the cerebro-spinal fluid is free, large numbers of red cells are liberated, and rapidly become diffused throughout the sub-arachnoid space. If, at the other extreme, the site of the hæmorrhage be in the lateral ventricle, the path of outflow through the iter is relatively narrow and the great majority of red cells are entangled in the clot. Such as do find their way to the basal cisterns rapidly diffuse throughout the great sub-arachnoid lake. Hence two of the characteristic features of the fluid obtained at lumbar puncture—the even admixture of the red cells, and the absence of coagulum.

The coloration of the supernatant fluid appears to be due to pigments derived from the red cells in process of destruction. This process of hæmatolysis is in its most active phase a few days after the initial effusion, and it is at this time therefore that the colour change is most remarkable. The tint is variable. In the earliest stages there may be very little or no coloration. The rose-yellow or brownish tint appears to coincide with the height of hæmatolysis in a large effusion, and the spectroscope shows the oxyhæmoglobin or hæmoglobin absorption bands; this colour yields later to a golden yellow

which gives the chemical reactions proper to bile pigments. In the case of a small effusion the colour may never be darker than golden yellow.

The microscopic changes which are to be observed in the sanguineous fluid have been carefully studied by Widal, Froin and others. In the earliest stage the number of white cells in proportion to the red cells is that met with in the blood. Later there is an increase in the white cells, at first especially of polymorphs and large mononuclears, subsequently of lymphocytes; and a small increase of lymphocytes may still be observed when the red cells have disappeared from the fluid, and this is clear and colourless.

Whereas the most conclusive evidence of sub-arachnoid hæmorrhage is afforded by the state of the cerebro-spinal fluid, this does not as a rule aid us in distinguishing the *source* of the hæmorrhage—whether it is of primary meningeal origin, or comes from an intracerebral effusion which has broken into the subarachnoid space.

There are, however, other clinical features which, in all but immediately fatal cases, serve, as a rule, to distinguish these two groups, the meningeal and the cerebro-meningeal hæmorrhages.

A cerebral hæmorrhage of any size as a rule gives rise to focal symptoms, usually a hemiplegia which is persistent. If the effusion ruptures into the meninges the signs of meningeal irritation will ensue as an additional symptom. If on the other hand there is rupture into one of the ventricles, the sequelæ consist of profound coma, together with generalized muscular rigidity and hyperpyrexia.

In primary meningeal hæmorrhage, on the other hand, signs of local cerebral damage are generally absent, or if present are slight and transient owing to the readiness with which the effusion finds an outlet into the sub-arachnoid lake. The symptoms in individual cases vary according to the situation, the suddenness, and especially the magnitude of the extravasation.

(1) At one extreme are cases in which the onset is apoplectiform, and death ensues rapidly from cerebral compression. In these instances, though lumbar puncture reveals the existence of sub-arachnoid hæmorrhage, it is impossible to distinguish the meningeal from the cerebro-meningeal hæmorrhage.

(2) In a second group of cases the onset may be sudden, with initial loss of consciousness and a variable period of coma due to cerebral compression, which passes off as the effusion becomes spread out, and reveals the signs of meningeal irritation. In this group the absence of any persistent signs of local cerebral destruction is valuable evidence in favour of meningeal rather than cerebro-meningeal hæmorrhage.

The following case illustrative of this group is one which I saw at Lambeth Hospital through the kindness of Dr. Bailey.

A labourer, aged 47, having previously enjoyed good health, had a sudden seizure with loss of consciousness. He was admitted a few days later (the history is incomplete) with the tentative diagnosis of encephalitis lethargica. He was then noted to be "very drowsy, speaks incoherently, plantar reflexes flexor, knee-jerks present, pupils react normally."

On the day following admission Dr. Stebbing noted that he was very drowsy but would answer questions with his eyes half-closed. He found weakness of both sixth nerves, but confirmed the absence of any other physical signs.

Cerebro-spinal fluid obtained the same day was examined by Dr. Perdrau, who reported as follows:—

"Fluid clear and brownish, no clotting on standing. Total protein, 1 per cent., sugar less than normal. Chlorides, 0·68 per cent.; cells, 69 per cubic millimetre, and 15 red blood cells per cubic millimetre. Differential count of whites: 95 per cent.

lymphocytes; 5 per cent. polymorphs. No plasma cells. Spectroscopic examination showed two absorption bands of a hæmoglobin derivative."

When I saw him on the following day, I found that he was conscious but drowsy with sighing respiration. There were no signs of local destruction of the central nervous system. He showed, however, definite neck stiffness, and a positive Kernig's sign. Ophthalmoscopic examination revealed neuro-retinitis of the typical albuminuric appearance together with one very large hæmorrhage near the left optic disc, and a few small hæmorrhages in the right fundus. The systolic blood-pressure was 190, and the urine contained a trace of albumin. The Wassermann reaction in the blood was negative.

The patient made a gradual but uninterrupted recovery, and left the hospital after two months in apparently good health. His systolic blood-pressure at that time was 210.

On discharge he resumed work for a month, but was unable to keep it up owing to general debility. Nine months later he was readmitted, having been struck down with apoplexy in the street. He was found on admission to be comatose, with a right hemiplegia, and died within twenty-four hours.

The autopsy revealed a recent large hæmorrhage occupying the greater part of the left hemisphere. No signs of any previous cerebral hæmorrhage were discovered. The arteries at the base of the brain were not carefully examined. The kidneys showed the characteristic changes of chronic Bright's disease.

I conclude that in this case the original illness was due to rupture of some small vessel at the base of the brain with subsequent healing. I was unfortunately not able to be present at the autopsy, or I should have made careful search for it.

It is difficult at first thought to understand how such a ruptured vessel could heal, especially as in this case it had to withstand a blood-pressure of 200. That arterial leakage into the sub-arachnoid space may be a recoverable condition is clearly proved by the number of persons with intracranial aneurysm who have survived such accidents for months or even years.

I take it that there are two factors in sub-arachnoid hæmorrhage which tend to promote recovery. First, the pressure of the effusion is evenly distributed throughout the whole sub-arachnoid space instead of being confined to the cranial contents as in an intracranial hæmorrhage, and the risk of death from acute cerebral compression is therefore lessened; and in the second place the fine meshes of the sub-arachnoid cavity promote coagulation.

(3) In the third group of cases the leakage is so gradual or so slight as to produce little or no cerebral compression, and the signs of meningeal irritation are unmasked. In these cases, even if the onset be sudden, consciousness may not be lost.

A case which I have published in the *Guy's Hospital Reports* serves to illustrate this group:—

Colonel E., aged 70, a single, retired officer of the Indian Medical Service, had led an active, busy life up to the date of the illness to be described.

On July 29, 1922, he was in his usual health. On the following morning, however, he complained to his landlady, on being called, that he had had a very bad night, with pain in the back of the neck. An hour or two later he was discovered lying in the bath-room in a dazed condition with a small scalp wound in the right parietal region.

He was conscious when found, and said that the pain in the back of his head had suddenly become intense, "something seemed to snap there," and he fell.

He was able to walk downstairs with support, went to bed, and stayed there for the next few days, though he got up to shave and wash. During this time he had a very foul, coated tongue; he was constipated and "a little light-headed" at times. Besides the pain in the neck, he complained of considerable pain in his thighs, so that,

for example, when he began to walk and bent his knees he would stop suddenly with the severity of it.

His condition slowly improved until August 10, when he had bacon and eggs for breakfast. He had had his bowels open and his tongue was clean.

At 12.15 a.m., on the 11th, he again sent for a medical friend, complaining of intense pain in the back of the neck, which was arched backwards. Subsequently he became gradually unconscious.

When I first saw him the same afternoon he was in a state of coma with sterterous breathing. He lay with his head turned a little to the right. Both eyes were open, but with the left lid a little lower than the right. The pupils were small—the left slightly the larger—and did not react to light. There was some external strabismus of the left eye. Slight weakness of the left face. The pulse was 48, the systolic blood-pressure 150 mm. Hg. The peripheral arteries to the feel, and the retinal vessels to the view, appeared to be in good condition.

On examination, it was noted that there was a definite backward rigidity of the neck. Kernig's sign was also present. He grimaced as if with pain when an attempt was made to carry the passive movement past a certain point.

In addition to this there was some rigidity of the limbs. The upper limbs were flexed at the elbows and pronated, the lower limbs fully extended, feet arched and toes pointing downwards. There were occasional purposive movements of the right arm.

The comatose condition of the patient made sensory examination impossible. As to the reflexes, none of the tendon-jerks could be obtained; both the plantar reflexes were extensor. He had retention of urine, and catheterization was necessary.

On August 12, he rallied in the early morning, became conscious, asked for water, and expressed a wish to pass urine, which he was able to do at will. At 10.30 a.m. he reeognized his friends and talked with them. At 3 p.m. he suddenly became unconscious again and died six hours later.

The post-mortem performed the next day was limited to the brain. On removal of the skull and incision of the dura, the subdural space at the vertex seemed to contain free blood, but this was small in quantity and may have escaped from an accidental rent in the arachnoid. At the base there was no doubt that the blood had escaped into the subdural space, for it was thickly clotted in both temporal fossæ after the brain with the lepto-meninges had been removed.

The sub-arachnoid space over the greater part of the hemispheres contained no blood, but at the base this was abundant, obscuring all the vessels and nerves, covering the under surface of pons and medulla and extending in a thick layer down the spinal canal as far as one could see. When the cervical cord was cut through for removal of the brain, there was a considerable flow of blood from the spinal sub-arachnoid space.

After removal, a stream of water was directed upon the clot at the base, and it was gradually wiped and teased away. About the right side of the pons and medulla it was especially adherent, and at the angle of junction of pons and medulla on the right side, close to the origin of the sixth nerve, was a hard, oval clot, the size of an almond. This was dislodged with difficulty, as it appeared to extend into the substance of the brain stem.

On cutting the hard clot across, I observed a small cavity in its centre, and this I am inclined to think represented the cavity of a minute aneurysm.

A sagittal section of the pons and medulla revealed a clot the size of a cherry-stone to the right of the midline, which was directly continuous through the rent in the surface with the hard clot already described. The clot within the nervous substance was of a soft jelly-like consistency, and clearly of a date later than that of the external clot. It seemed, therefore, as if the hæmorrhage into the medulla had been from the surface inwards rather than in the opposite direction. Unfortunately the suspected aneurysm and adjacent parts, which were needed for further examination were, by an oversight, replaced within the cranium and lost.

The rest of the brain was of normal size and configuration, and on section showed no abnormality.

The cerebral arteries on the whole were in a very good condition, with the exception

of isolated plaques of atheroma in those of medium size, and extreme calcareous rigidity of the carotids.

In a second case, related in the same paper, the symptoms were very similar, but led to recovery, the evidence of the sub-arachnoid hæmorrhage being obtained from lumbar puncture.

This patient, a nursing sister, aged 52, was also able, after recovery, to give a vivid account of the onset of her illness.

Having previously been in her usual health, she suddenly felt vaguely ill while at lunch, and went to her room. "Then suddenly there was a whirling feeling at the base of my skull, and something seemed to snap."

She was found on the floor in a semi-conscious state and put to bed. Subsequently she complained of severe headache and pain in the back of the neck. When I saw her on the fifth day of her illness she presented considerable neck stiffness. The plantar responses were on the right doubtful, on the left occasionally extensor. Lumbar puncture revealed evenly blood-stained cerebro-spinal fluid. She made a gradual recovery.

The evidences of meningeal hæmorrhage, therefore, in addition to the state of the cerebro-spinal fluid, are the signs of acute meningeal irritation—headache, pain or stiffness in the neck and limbs, a positive Kernig's sign, and a dazed, irritable, mental condition. These signs of meningeal irritation may be preceded in severe cases by those of cerebral compression, or if the hæmorrhage be profuse, death may ensue from compression in the first stage.

There are two further clinical phenomena which may be observed in sub-arachnoid hæmorrhage and deserve separate consideration.

One is the occurrence of fever which is usually present in the first ten days, and is at its highest during the phase of most active hæmatolysis, with which it is doubtless associated.

The temperature chart shown is from a case which I shall refer to later, and appears, from a consideration of other cases reported, to be somewhat typical.

The association of pyrexia with signs of acute meningeal irritation has, it appears, frequently given rise to the suspicion of an infective meningitis, and this mistake in diagnosis has probably been committed more frequently than is discernible from the literature, the blood obtained at lumbar puncture being usually set down to an accidental cause. This hæmatolytic fever is analogous to that seen in other instances of bleeding into serous cavities with subsequent absorption.

The other phenomenon occasionally to be observed in meningeal hæmorrhage is the occurrence of subretinal hæmorrhages.

I have observed these in one case which I have reported, that of a woman aged 52, the subject of cerebral arteriosclerosis, who died from rupture of a small aneurysm of the right internal carotid artery within the cavernous sinus. In this case both discs were slightly œdematous, and in the left fundus were several large subretinal accumulations of blood, one of which partly obscured the edge of the optic disc. It was noted that these had not the ragged outline usually seen in large retinal hæmorrhages.

At post-mortem on the following day widespread sub-arachnoid hæmorrhage was discovered which extended into the sheaths of both optic nerves.

I have met with two similar observations, both in cases of ruptured aneurysm. The earliest is that recorded by Sir William Hale-White in 1895.[1]

His case was that of a labourer, aged 21, who had an aneurysm of the right internal carotid just before its termination, presumably of congenital origin, since there was no other evidence of disease.

After an initial seizure with brief recovery he developed the characteristic symptoms of progressive meningeal hæmorrhage.

[1] *Trans. Clin. Soc. Lond.*, 1895, xxviii, p. 7.

On examination of the fundi "there was no optic neuritis, nor atrophy, but on the outer side of the right optic disc was a large prominent, dark brick-red, sub-retinal swelling. This encroached a little on the disc and was four times the size of it. It was thought to be a subretinal hæmorrhage."

At the post-mortem "there was a large amount of clot on the under-surface of the brain, which extended to the fourth ventricle and was ⅛ in. thick in the sub-arachnoid space all down the spine. The hæmorrhage had passed forwards in the sheaths of the optic nerves, which were much distended with blood-clot, and ultimately tore its way forwards under the retina."

The other instance is recorded by Doubler and Marlow. Here again the cause was an intracranial aneurysm of the internal carotid artery which had apparently ruptured.

The patient was a woman of 32, previously in good health, who died ten and a half hours after the onset of her illness. She was admitted in a state of semi-consciousness with neck stiffness and absent tendon-jerks. Lumbar puncture revealed a heavily blood-stained fluid.

"At the first ophthalmoscopic examination of the right eye, a large, red, apparently fresh hæmorrhage was seen located on the disc, partly obscuring its upper half. There were a few smaller, peripheral flame-shaped hæmorrhages not impinging upon the disc or any blood-vessel. . . . Both fundi were examined from time to time. The large hæmorrhage on the disc in the right eye grew until the disc was completely obscured. It partly filled the nasal side of the fundus, and appeared to be extending into the vitreous. At one examination it seemed as though the hæmorrhage could be seen to undergo enlargement."

At the autopsy both optic nerves and the posterior parts of the eyeballs were removed.

Cross section of the optic nerves showed the sub-arachnoid and subdural sheaths to be distended by blood which extended as far, anteriorly, as the lamina cribrosa. At no place did this hæmorrhage invade the nerve itself. The hæmorrhage upon the right papilla appeared to be a direct extension from the nerve sheath, through the lamina cribrosa.

Other observations of a similar nature have probably been made. I shall hope to hear of some this evening.

The fact that in the three cases which I have related the hæmorrhage originated in each instance from aneurysm of the internal carotid is probably not without significance. Both the size of the vessel and its situation would favour a pressure of fluid blood within the optic nerve sheath sufficient to cause extravasation into the sub-retinal spaces.

It would appear then that for the recognition of sub-arachnoid hæmorrhage at the bedside we have at our disposal reliable clinical data, and when to this is added an opportunity of examining the cerebro-spinal fluid, the true diagnosis may often be arrived at with reasonable certainty. When, however, we come to inquire more particularly into the exact sources and pathological causes of the hæmorrhage, we are confronted with greater difficulties.

It is natural first to suspect those causes which commonly lead to vascular degeneration or rupture, whether in the central nervous system or elsewhere, and of these the most important is arteriosclerosis. In this disease degeneration of the media, together with a rise of blood-pressure, may result either in direct rupture of the vessel, or in the formation of an aneurysm which in its turn commonly bursts. Examples of both these accidents I have already quoted. Actually, such cases must be of comparatively frequent occurrence, yet the literature of primary sub-arachnoid hæmorrhage is scanty, probably because most cases are put down as cerebral hæmorrhage without sufficient inquiry.

From papers directly dealing with sub-arachnoid hæmorrhage I have been able to collect descriptions of sixty-three cases in which adequate data are available, and to these I can add seven observations of my own. I have included only those in which lumbar puncture or post-mortem examination has afforded clear proof of the diagnosis, and I have been careful to exclude cases in which the meningeal hæmorrhage appeared to be secondary to an intracerebral effusion.

Of these seventy cases twenty-two are definitely recorded as having shown signs of generalized arteriosclerosis with or without chronic nephritis. Five of these recovered from their attack of sub-arachnoid hæmorrhage, seventeen died. Of these, in six a ruptured artery was found—five times at the base of the brain, once in the lumbar region. In three the source was a ruptured aneurysm, while in the remaining eight the source was not found. With regard to this latter point, it must be remembered that great care and patience are required to discover amidst widely-spread masses of clot a minute rent in the wall of an artery, for instance of the size of the anterior cerebral, and even a small aneurysm is easily missed.

Therefore, I think we may fairly assume that primary sub-arachnoid hæmorrhage occurring in an old person with diseased vessels is due to the same causes which commonly determine *intra*-cerebral hæmorrhage.

Amongst the remainder of my series of seventy cases of sub-arachnoid hæmorrhage there are twenty-one others in which the accident could be attributed to a reasonable cause or was associated with pathological changes elsewhere in the body. Thus, there is a case of hæmophilia, another of pernicious anæmia, another of rupture of a vessel in the wall of a cyst. There are also six cases in which the patient is either recorded to have had positive Wassermann reactions at the time, or was known to have suffered from syphilis a few years previously. Only one of these patients died, and in this instance no definite source for the hæmorrhage was found at autopsy. The remainder improved under anti-syphilitic treatment.

The pathological process at work in these cases is probably that which Turnbull has described as a somewhat uncommon form of syphilitic arteritis. As he has pointed out: "When the smaller muscular and elastic arteries are the seat of syphilitic inflammation, endarteritis is usually a marked feature, the lumen becoming almost obliterated." However "in some cases of intense inflammation with necrosis the adventitia and media become greatly weakened before the intima has greatly thickened." And it is in such cases that rupture is likely to occur.

Five instances of sub-arachnoid hæmorrhage occurred in patients addicted to chronic alcoholism, a condition in which vascular degeneration frequently occurs. In two of these cases there were associated hæmorrhages in other parts of the body.

In three cases the symptoms of meningeal irritation are said to have developed acutely in persons exposed to the rays of a tropical or sub-tropical sun. All three recovered. Sir William Willcox has published observations of extreme congestion of the brain and meninges in fatal cases of heat stroke seen in Mesopotamia during the war, and of other cases in which recovery was associated with the signs of cerebral lesions presumably due to hæmorrhage, but I am not aware of any recent instances in which a blood-stained cerebro-spinal fluid has been observed during life or sub-arachnoid hæmorrhage demonstrated at autopsy.

Finally, there is a group of five cases in which the hæmorrhage was proved post mortem to have originated from an aneurysm at the base of the brain in

the absence of any evidence of arterial degeneration or other disease. In each of these cases the patient was young. In two instances I have been unable to obtain access to the original descriptions. In the other three the ages were 36, 32 and 24.

The last of these I was able to bring to light quite recently. The case was that of a young man, aged 24, who came into Guy's Hospital last September, and died a few hours after admission. He was in a state of coma when first seen, and is stated to have had epileptiform convulsions while under observation. No previous history was available. Just before death he appears to have had a small amount of sugar in his urine. The provisional clinical diagnosis made was status epilepticus. At the post mortem there was abundance of blood in the sub-arachnoid spaces at the base of the brain with extension upwards into the Sylvian fissures and the neighbouring sulci. The rest of the bodily organs were those of a healthy young man. The diagnosis suggested was that in the course of an epileptic convulsion one of the cerebral veins had given way under the increased pressure.

Having obtained permission to examine the brain and with the possibility of an aneurysm before my eyes, I proceeded to dissect the main arteries out of the clot in which they were imbedded, working upwards from the circle of Willis, and discovered a small thin-walled aneurysm at the first point of bifurcation of the right anterior cerebral. The aneurysm lies actually in the apex of the Y formed by the two branches, which both appear to take origin from it. Its free wall, which is of the thickness of a sheet of notepaper, has ruptured.

Here, then, is a case in which the cause of a fatal meningeal hæmorrhage might have remained obscure had not the aneurysm been carefully looked for. The remarks of Sir William Gull, written in 1859, are so much to the point in this connexion that I may perhaps be forgiven for quoting them :—

" Aneurism of the cerebral vessels has been regarded as a disease of extreme rarity, and judging by the scanty records of it, we should conclude that the opinion was true. This apparent rarity, however, like all negative conclusions, is doubtful, and I think there is the more reason to suspect it as only apparent and due to careless inquiry, since the discovery of these cases has been much more frequent during the last ten years. There are several reasons why intracranial aneurism is likely to be overlooked. First of all, as here hinted at, it has not been looked for, and it is notorious that the eye can see only that it brings with it the aptitude to see. Again, when death occurs from rupture of the sac, recent coagula may so imbed and conceal it that unless strictly looked for it will not be found, for the sac is often small, and thin and transparent, except at the point of rupture. Further, also, when death has taken place from changes around the aneurism, as by pressure or softening, the sac itself may present such appearances that unless a minute dissection be made of it, its true nature may not be discovered. Whenever young persons die with symptoms of ingravescent apoplexy, and after death large effusion of blood is found, especially if the effusion be over the surface of the brain in the meshes of the pia mater, the presence of an aneurism is probable." [1]

In the case which I have just related the cause of the aneurysm was almost certainly that which has been alluded to by Turnbull as an " inherent weakness due to a congenital abnormality in the structure of the arteries at their points of junction."

Turnbull has recorded an instance of one of these aneurysms found at the junction of left anterior cerebral with anterior communicating, in a child aged 1 year and 7 months who died of broncho-pneumonia complicating gastro-enteritis.

The late Dr. Fearnsides, adding forty-four new cases to the literature of intracranial aneurysm in 1916,[2] found that of thirty-one cases in which non-inflammatory degeneration of the media was the cause, in fifteen (or 48·4 per cent.) no cardio-vascular hypertrophy was present.

[1] *Guy's Hosp. Rep.*, 3rd ser., 1859, v, p. 281.
[2] *Brain*, 1916, xxxix, pp. 244-296.

Whilst the literature of sub-arachnoid hæmorrhage is scanty that of intracranial aneurysm is relatively abundant. Beadles, in 1907, was able to collect 555 cases including 114 of his own, and many have since been published. The group of congenital origin has not been recognized until recent years although the fact was generally recognized that intracranial aneurysms might be found in young and healthy people. If Fearnsides' figures prove a reliable index then congenital aneurysms must be of not infrequent occurrence, for they were found fifteen times in a series of 5,432 consecutive examinations of the head—one in every 362 cases.

Moreover, in many cases of intracranial aneurysm there occur multiple leakages of blood due to partial rupture of the sac, before the final breach occurs. (This was so in 41·9 per cent. of Fearnsides' cases.) I suspect, therefore, that in many of the cases which have been recorded as obscure sub-arachnoid hæmorrhage the cause has been an unrevealed congenital aneurysm. When the patient has died this may well have escaped detection if the possibility of its existence was not entertained in the mind of the examiner.

In those cases, on the other hand, in which the patient has recovered from the first attack, he may appear to have regained his normal health for months or even years before further leakage takes place. In such cases, therefore, the possibility of a congenital aneurysm cannot be excluded without a history of prolonged subsequent observation, and in none of the published instances is this forthcoming.

I have published in the *Guy's Hospital Reports* as a probable case of congenital aneurysm the record of a man, now aged 20, who has suffered from three attacks of sub-arachnoid hæmorrhage.

He was first admitted to Guy's, at the age of 12, with the symptoms of acute meningitis. Lumbar puncture on the fifth day of his illness revealed a fluid stated to be normal save for a slight admixture of blood. He made a rapid and uneventful recovery. He was readmitted at the age of 17 for a similar illness. On this occasion the cerebro-spinal fluid was obtained on the sixth and ninth days of the illness, and showed the appearances characteristic of sub-arachnoid hæmorrhage. The third attack occurred in January, 1923, at the age of 19, when he first came under my observation. In addition to the signs of acute meningeal irritation, and the presence of much blood in the cerebro-spinal fluid, he showed on this occasion some swelling of the discs, a right third nerve palsy, and a left-sided hemiparesis. He made a rapid recovery, but still showed some left hemiparesis and complained of occasional right frontal headaches when last seen.

Actually, on looking through the seventy cases which I have collected under the heading of sub-arachnoid hæmorrhage there appear to be twenty-seven in which no satisfactory cause could be assigned for the accident. Of these seven died, and the autopsy notes record the existence of profuse sub-arachnoid hæmorrhage (as a rule basal) without the discovery of any ruptured vessel. The remaining twenty patients recovered and of these the after-histories are available only in three cases of my own.

It is noteworthy that the age incidence in the obscure group is relatively low compared with other groups. The youngest patient was 10, the oldest 56, with an average age of 28.

The distribution is shown more clearly in tabular form:—

Between	the	ages	of	10	to	20	...	...	6 cases
,,	,,	,,	,,	20	,,	30	...	...	11 ,,
,,	,,	,,	,,	30	,,	40	...	...	2 ,,
,,	,,	,,	,,	40	,,	50	...	...	2 ,,
,,	,,	,,	,,	50	,,	60	...	...	4 ,,

In Fearnsides' fifteen cases of aneurysm which appeared to belong to the congenital group the ages varied from 19 to 53 with an average age of 38.

These figures, as far as they go, may be taken as evidence in favour of the view which I have already put forward that in a number of the obscure cases the cause of sub-arachnoid hæmorrhage has been an unrecognized aneurysm of congenital origin.

Another theory has recently been put forward by Goldflam in explanation of certain of the obscure cases of sub-arachnoid hæmorrhage. He notes, as others have done, that many of the patients attacked are young persons in normal health at the time of onset. Taking this in conjunction with the negative post-mortem appearances in certain fatal cases, he suggests that the cause may be a functional disturbance of vasomotor control analogous to that which is said to occur in migraine, Raynaud's disease and erythromelalgia. His thesis appears to be that in the stage of active hyperæmia, which follows that of abnormal vaso-constriction, capillary oozing may take place upon the surface of the brain. In support of his theory he refers to thirteen cases of sub-arachnoid hæmorrhage personally observed, in no less than five of which the patients suffered from true migraine.

Unfortunately no detailed account of these cases is given, and this largely detracts from the value of the paper. Goldflam also appears to be unaware of the congenital group of aneurysms, for he rules out the possibility of aneurysm in ten of his cases on the ground that they were all under 30 years of age and presented no signs of vascular disease.

In the seventy cases which form the basis of the present paper I have searched carefully for a history of migraine in every instance, but have discovered mention of it only in two cases.

One is the case recorded by Ingvar of a man aged 34 who is said to have suffered from typical migraine for four or five years. He suffered also from chronic nephritis with a blood-pressure of 165. The case terminated fatally and although no actual defect was actually found, microscopic examination of the cerebral arteries showed widespread degenerative changes.

The other case is one which came under my own observation, and it is, I think, sufficiently remarkable to deserve your comments.

The patient is a man now aged 37, who gives the following history: His father is said to have died suddenly at the age of 42 from "hæmorrhage of the gums." I can obtain no details of this illness. He had not previously suffered from bleeding, and there is no evidence to suggest a hæmorrhagic diathesis.

The patient has since the age of 11 suffered from attacks which he describes as being of two separate varieties.

The one variety is clearly of an epileptic nature. There is an aura of pins and needles and twitching, sometimes commencing in the hand, sometimes in the foot, but always on the left side. This is followed by clonic spasms of the left arm and leg. He then loses consciousness. His wife states that loss of consciousness occurs as the movements begin to involve the right side of the body. He enters into a tonic state with cyanosis, followed by generalized clonic movements, and passes out of coma into a deep sleep, from which he wakes in about three hours with severe headache.

The other variety of attack he describes as follows:—

He will perhaps be walking in the street when he becomes aware of what he calls "a spot" obscuring his vision on the left side. This spot looks to him like an irregular watery patch always on the move, which gradually increases in size until his whole vision appears blurred, as if he were looking through a mist. At the same time he sometimes experiences a feeling of pins and needles in the left hand, face and foot. There is never any nausea, vomiting or headache. He does not lose consciousness and is able to carry on with his work as long as this does not involve reading, for which his vision becomes too dim. The whole attack lasts a few minutes.

These attacks, he says, have no relation whatever to the epileptic attacks, and do not alternate with them.

On May 30, 1923, he had a typical epileptic attack in a cinema. During the following week he felt poorly, and complained of a muddled head, but continued at work. On June 6 he played tennis for three hours. At 7 p.m. he went into his garage and cranked up his car. He then came out complaining of a severe pain in the back of the neck. His wife noticed a little twitching of the left hand and foot, and expected him to have one of his usual fits. Instead of this he continued to complain of severe headache, and pain in the back of the neck, and he vomited. The pain and vomiting continued for twenty-four hours and his medical man administered morphia. He remained in bed for ten days, and on getting about again he complained of pain in the bottom of the back. This was shortly followed by a recurrence of the headache, vomiting and neck pain. Lumbar puncture was performed by his medical attendant, and a pinkish fluid which contained no organisms is said to have been withdrawn.

As his condition did not improve he was sent into Guy's Hospital two days later. On admission he was semi-conscious, his temperature was 100°, he presented head retraction and a positive Kernig's sign; no other abnormal signs.

Lumbar puncture yielded blood-stained fluid. In the tube was a considerable sediment of red cells: there was no clot; the supernatant fluid was yellow and gave a negative response with Van den Bergh's direct reaction, and a positive result with the indirect test (the reaction for altered blood pigment).

The protein was 0·09 per cent. The Wassermann reaction was negative in blood and spinal fluid.

He made a gradual recovery and when seen six months later was in good health. He has had several migrainous attacks lately, but no attack of epilepsy since his acute illness.

If there is any theoretical basis for Goldflam's hypothesis here at any rate is a case in which his suggested interpretation would be welcome.

There are many points in this case which call for comment, but time forbids my dealing with them.

In general the prognosis and treatment in a case of sub-arachnoid hæmorrhage must depend upon the primary cause. The question of immediate practical importance once the diagnosis has been made is whether or not to attempt drainage of the sub-arachnoid space by means of lumbar puncture. This has been advocated by many authorities as a routine practice. On theoretical grounds, however, it would appear that due consideration should be given to the individual case with especial regard both to the probable source of the hæmorrhage and its amount.

There is no doubt that in sub-arachnoid hæmorrhage death may occur rapidly from cerebral compression. Drainage of the effusion by means of lumbar or cistern puncture in these cases would seem to be the most hopeful measure of relief. On the other hand, there is a danger in this procedure lest diminution of external pressure upon the point of rupture may lead to fresh bleeding. Cases have been recorded of intracranial aneurysm in which death has occurred immediately after lumbar puncture from this cause. In the early stages of sub-arachnoid hæmorrhage, therefore, the physician must be guided by the presence and severity of signs of cerebral compression.

If life appears to be threatened from this cause the indication for therapeutic puncture is urgent. Yet even to this rule there must be an exception. If the source of sub-arachnoid hæmorrhage be an intraventricular effusion just those conditions are likely to develop which we often see in the case of cerebral tumour—the formation of a pressure cone of cerebellum upon the roof of the medulla, in which case lumbar puncture may lead at once to a fatal issue.

Clinical evidence of intraventricular hæmorrhage should, therefore, be contra-indication for this procedure.

In the later stages when hæmorrhage appears to have ceased, but the

patient is suffering from the effects of the meningeal reaction to the clot, lumbar puncture may be employed to relieve pain and accelerate recovery. Yet even at this stage I feel that it should be used sparingly, with a watch upon the pressure of the escaping fluid and due regard to the probable existence of a recently healed scar only separating the arterial blood stream from the sub-arachnoid space.

In conclusion I would remind you that I have only been able to find in English text-books and journals scant reference to my subject, and would express the hope that some of you may be able to repair this omission both by directing attention to further sources of information and by drawing upon your personal experiences of spontaneous sub-arachnoid hæmorrhage.

DISCUSSION.

Dr. WILFRED HARRIS said he was indebted to Dr. Symonds for his suggestion that the bleeding in some of these cases was due to the formation of congenital aneurysms, but he considered that venous hæmorrhage was an important cause, especially in young subjects. He described the case of a man who lived for fourteen days with a very large collection of blood under the arachnoid. Another interesting example of these was the case of a man who during coitus had a sudden intense headache, and who showed the characteristic picture in the cerebro-spinal fluid.

Mr. LESLIE PATON said that there was one possible cause of sub-arachnoid hæmorrhage to which he would call Dr. Symonds' attention, and that was scurvy. Freud, in a paper published in the *Wiener med. Wochenschrift*, in 1884,[1] described a case of scurvy in which there were bleedings in the subdural space of the brain, in both optic nerve sheaths and all over the inner surface of the retina, and into the vitreous of the left eye ; in the right eye there were only small retinal hæmorrhages.

In dealing with these cases of sub-arachnoid hæmorrhages and their ophthalmic complications it was essential to bear in mind that many of the conditions which might cause sub-arachnoid hæmorrhage might be acting in the retinal vessels, and might produce hæmorrhages and changes in the retina which were not necessarily subordinate results of the sub-arachnoid hæmorrhage. In the cases in which the cerebral blood-vessels were diseased the retinal vessels were probably also diseased, and retinal hæmorrhages might be caused by disease of the retinal vessels. On the other hand, there was little doubt that occasionally sub-arachnoid hæmorrhages did produce direct ophthalmoscopic changes. These changes might be brought about in two ways, first by direct hæmorrhage into the nerve sheath, producing pressure in the nerve sheath and papillœdema. In this case, the pressure was accompanied by loss of sight, and usually the pupil reaction was lost. In the second type of case the sub-arachnoid hæmorrhage was due to the presence of a hæmatoma acting like a cerebral tumour, and giving rise to increased intracranial pressure. The comparative rarity of papillœdema in cerebral hæmorrhages was probably due to two factors. If the hæmorrhage was large enough to produce much increase in intracranial tension on its own account, the patient was usually dead before the increased intracranial tension had had time to produce any effect on the disc head. On the other hand, if the hæmorrhage was small, it was often absorbed fairly rapidly, without producing more than a temporary rise in intracranial pressure. The type of case in which a cerebral hæmorrhage was most likely to produce a papillœdema was that in which the hæmorrhage became encysted. A case of this kind, which he (Mr. Paton) saw more than twenty years ago, was under Dr. Taylor's care at the National Hospital. A large, encysted hæmatoma extended from the occipital region behind to the frontal region in front, entirely covering the parietal region. Its removal was followed by complete subsidence of the papillœdema with retention of perfect vision.

The direct appearance of hæmorrhage through the retina, making its way through the lamina cribrosa and appearing in the eye, was very rarely seen, but he could record two cases in which there had been large sub-hyaloid hæmorrhages into the blood obviously coming down the lymph channels beside the central vessels and in the

[1] Wien. med. Wochenschr., 1884, xxxiv, pp. 244; 276.

subpial zone, and forming large sub-hyaloid hæmorrhages, which in the case that survived ultimately burst into the vitreous.

Dr. F. PARKES WEBER supposed that the term "congenital aneurysm" did not signify that the aneurysm was really congenital, but only that it developed in a congenitally imperfect or predisposed artery. The rare cases of non-syphilitic aneurysms, not of inflammatory nature, occurring in hypoplastic aortas might be mentioned in that connexion. In regard to Goldflam's suggestion that migraine might be an exciting cause of sub-arachnoid or cerebral hæmorrhage—Dr. Weber remembered the case of a man, aged 33 years, subject to attacks of migraine and temporary blindness, who had an intra-ocular hæmorrhage (into the vitreous of the right eye), which, it was suggested, might have been due to extreme venous hyperæmia during the period of arterial relaxation following migrainous preliminary arteriospasm. In another case (a woman, aged 52 years), subject to migraine-like attacks, detachment of the retina had occurred in the right eye. No allusion had been made to the possibility of spontaneous cerebral or sub-arachnoid hæmorrhage in young and apparently healthy subjects being occasionally due to congenital angiomatous or nævoid telangiectatic conditions. But that explanation for the recurrent attacks of hæmorrhage in one of the cases referred to seemed at least as probable as the "congenital aneurysm" explanation. In a recent elaborate work Dr. O. P. Reuterwall[1] recorded his careful examination of the basilar artery in eighty-seven post-mortem examinations; in seven of these he found transverse rents in the internal elastic lamina which during life had undergone connective-tissue repair. Similar conditions would probably have been found in other cerebral and meningeal arteries had they been carefully examined. As these rents tended to be multiple, the internal elastic layer was probably peculiarly "fragile" in such cases. The ages in the seven positive cases varied between 38 and 70 years, and, though in all of them there were arterial atheromatous changes present, the rents had occurred at almost healthy or only slightly diseased sites. Obviously, the same causes (probably blood-pressure strain, slight local disease and a condition of "fragility") which produced the rents in the internal elastic lamina might, in some cases, give rise to complete rupture and hæmorrhage or to the formation of an aneurysm. Reuterwall's work threw light on the occasional occurrence of cerebral hæmorrhage and cerebral aneurysm in young and apparently healthy individuals apart from syphilis and local active inflammation—that is to say, in arteries only slightly diseased.

Dr. W. J. ADIE referred to four cases which had been under his observation, in two of which the typical clinical picture of cerebro-spinal fever was present. Two other cases had occurred in young children, beginning with a very sudden and intense headache. He mentioned the observation of Guillain as to the occurrence of a massive albuminuria in some cases. Mental changes might persist for a long time. He did not think that ruptured aneurysms could explain all the cases.

Mr. F. A. WILLIAMSON-NOBLE described a case in which the cerebro-spinal fluid was deeply blood-stained, and the condition suggested an acute cerebro-spinal fever, but the case on further observation proved to be an example of anthrax meningitis.

Dr. J. G. GREENFIELD referred to the question of the colour of the spinal fluid in these cases. He had not seen the one pink colour which Dr. Symonds had mentioned, but colours varying from brown to orange or yellow. The nature of the pigment was uncertain, but the amount of bile pigment was very small.

Dr. JAMES COLLIER (President) said that with regard to the occurrence of congenital aneurysms, the late Dr. Trevor had a collection of eighteen such cases at St. George's Hospital, in which sub-arachnoid bleeding had taken place. In his experiences these cases had varied in age from 18 to 72. He (Dr. Collier) laid stress on the occurrence of convulsions in certain cases; in two examples these had occurred as late as the twenty-first day after the onset of the first symptoms. Many of his cases had at first been treated as severe rheumatism of the neck. He had employed spinal puncture freely and repeatedly as a therapeutic agent with great relief to the patients, and he thought that the danger of this form of treatment was exaggerated.

Dr. SYMONDS replied.

[1] O. P. Reuterwall, "Ueber bindegewebig geheilte Risse der Elastica interna der Arteria basilaris," Stockholm, 1923.